WHAT IS CLAIMED IS:

1. A non-human transgenic mammal, the cells of which comprise at least one non-functional endogenous LXRα allele.

2. The non-human transgenic mammal of claim 1, wherein said cells comprise two non-functional endogenous LXRα alleles.

3. The non-human transgenic mammal of claim 1, wherein said mammal is selected from the group consisting of mouse, rat, hamster, guinea pig, rabbit, cow, and sheep.

4. The non-human transgenic mammal of claim 1, wherein said non-functional LXRα allele contains an interruption in the LXRα coding sequence.

5. The non-human transgenic mammal of claim 2, wherein said non-functional LXRα alleles both contain an interruption in the LXRα coding sequences.

6. The non-human transgenic mammal of claim 1, wherein said non-functional LXRα allele contains a nonsense mutation that truncates the LXRα product.

7. The non-human transgenic mammal of claim 2, wherein said non-functional LXRα alleles both contain a nonsense mutation that truncates the LXRα products.

8. The non-human transgenic mammal of claim 1, wherein said non-functional LXRα allele contains a deletion of LXRα coding sequences.

9. The non-human transgenic mammal of claim 2, wherein said non-functional LXRα alleles both contain a deletion of LXRα coding sequences.

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10. The non-human transgenic mammal of claim 1, wherein said non-functional allele contains an alteration in the regulatory region of the LXRα gene.

11 The non-human transgenic mammal of claim 2, wherein said non-functional LXRα alleles both contain an alteration in the regulatory region of the LXRαs.

12. The non-human transgenic mammal of claim 10, wherein said alteration comprises substitution of an inducible/repressable promoter for the endogenous LXRα promoter.

13. The non-human transgenic mammal of claim 11, wherein said alterations comprise substitution of inducible/repressable promoters for both of the endogenous LXRα promoters.

14. The non-human transgenic mammal of claim 1, wherein cells of said mammal further comprise an exogenous selectable marker gene under the control of a promoter active in at least one cell type of said mammal.

15. A method for screening an RXR agonist or LXRα agonist candidate substance for the ability to increase bile acid synthesis comprising:

(a) providing a cell;

(b) contacting said cell with said candidate substance; and

(c) monitoring a bile acid-related phenotype of said cell,

wherein an increase in said bile acid-related phenotype in said cell treated with said candidate substance, as compared to a similar cell not treated with said candidate substance, indicates that said candidate substance increases bile acid synthesis.

16. The method of claim 15, wherein said cell is a liver cell.

17. The method of claim 15, wherein said bile acid-related phenotype is expression of a gene involved in bile acid synthesis.

18. The method of claim 17, wherein said gene is *Cyp7a*.

19. The method of claim 15, wherein said candidate substance is an RXR agonist.

20. The method of claim 15, wherein said RXR agonist is a rexinoid.

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21. A method for screening a candidate substance for the ability to reduce cholesterol levels in a mammal comprising:

(a) providing a non-human transgenic mammal, the cells of which comprise at least one non-functional endogenous LXRα allele;

(b) treating said mammal with said candidate substance; and

(c) monitoring a cholesterol-related phenotype in said mammal,

wherein a reduction in said cholesterol-related phenotype in mammals treated with said candidate substance, as compared to a similar mammal not treated with said candidate substance, indicates that said candidate substance reduces cholesterol levels.

22. The method of claim 21, wherein said mammal is selected from the group consisting of mouse, rat, hamster, guinea pig, rabbit, cow, and sheep.

23. The method of claim 21, wherein said phenotype is cholesterol absorption, circulating cholesterol, hepatic cholesterol, hepatomegaly, atherosclerosis, cardiac

failure, cardiac (atrophy/hypertropy), activity level, survival, cancer, reproduction, immune function, skin disease, cognitive function, and adrenal function.

24. The method of claim 21, wherein said mammal is maintained on a high cholesterol diet.

25. The method of claim 21, wherein said mammal further is treated with an agent that blocks cholesterol biosynthesis.

26. The method of claim 21, wherein said cells comprise two non-functional endogenous LXRα alleles.

27. A method for screening a candidate substance for the ability to increase bile acid synthesis in a mammal comprising:

(a) providing a non-human transgenic mammal, the cells of which comprise at least one non-functional endogenous LXRα allele;

(b) treating said mammal with said candidate substance; and

(c) monitoring a bile acid-related phenotype in said mammal

wherein an increase in said bile acid-related phenotype in mammals treated with said candidate substance, as compared to a similar mammal not treated with said candidate substance, indicates that said candidate substance increases bile acid synthesis.

28. The method of claim 27, wherein said mammal is selected from the group consisting of mouse, rat, hamster, guinea pig, rabbit, cow, and sheep.

29. The method of claim 27, wherein said bile acid-related phenotype is selected from the group consisting of cholesterol level, Cyp7a synthesis, fecal bile acid excretion, bile acid pool size and bile acid composition.

30. A method for screening a rexinoid for the ability to inhibit cholesterol absorption by an intestinal cell comprising:

(a) providing an intestinal cell;

(b) treating said cell with said rexinoid; and

(c) monitoring cholesterol absorption by said cell,

wherein a reduction in cholesterol absorption by said cell treated with said rexinoid, as compared to a similar cell not treated with said rexinoid, indicates that said rexinoid is an inhibitor of cholesterol absorption.

31. The method of claim 30, wherein said cell is an duodenal cell.

32. The method of claim 30, wherein said cell is located in a mammal.

33. The method of claim 30, further comprising comparing the effect of said candidate substance on cholesterol absorption on a cell comprising one or two non-functional endogenous LXRα alleles.

34. A method of reducing cholesterol levels in a mammal comprising the step of treating said mammal with an RXR agonist.

35. The method of claim 34, wherein said agonist is a rexinoid.

36. The method of claim 34, further comprising treating said mammal with an agent that inhibits cholesterol biosynthesis.

37. The method of claim 35, wherein said agent is HMG CoA reductase inhibitor.

38. The method of claim 34, wherein said mammal is a human.

39. The method of claim 34, further comprising stimulating bile acid synthesis in said mammal.

40. The method of claim 34, further comprising reducing cholesterol intake by said mammal.

41. A method for inhibiting cholesterol absorption in a mammal comprising treating said mammal with said with an RXR agonist.

42. The method of claim 41, wherein said agonist is a rexinoid.

43. The method of claim 41, wherein said mammal is a human.

44. A transgenic cell which comprises at least one non-functional endogenous LXRα allele.

45. The transgenic cell of claim 44, wherein said cell comprises two non-functional endogenous LXRα alleles.

46. A rexinoid compound that inhibits cholesterol absorption, identified by a process comprising:

(a) providing an intestinal cell;

(b) treating said cell with said rexinoid; and

(c) monitoring cholesterol absorption by said cell,

wherein a reduction in cholesterol absorption by said cell treated with said rexinoid, as compared to a similar cell not treated with said rexinoid, identified said rexinoid as an inhibitor of cholesterol absorption.

47. A rexinoid compound that inhibits cholesterol absorption, produced by a process comprising:

(a) providing an intestinal cell;

(b) treating said cell with said rexinoid;

(c) monitoring cholesterol absorption by said cell, wherein a reduction in cholesterol absorption by said cell treated with said rexinoid, as compared to a similar cell not treated with said rexinoid, identified said rexinoid as an inhibitor of cholesterol absorption; and

(d) producing said rexinoid compound.

48. A method of screening for a modulator of ABC1 expression comprising:

(a) providing a cell expressing an RXR;

(b) contacting said cell with a rexinoid and a candidate substance; and

(c) determining the expression of ABC1 in said cell,

wherein a change in expression of ABC1, as compared to a cell of step (b), indicates that said candidate substance is a modulator of ABC1 expression.

49. The method of claim 48, wherein ABC1 expression is measured by RNA analysis.

50. The method of claim 49, wherein said RNA analysis is Northern analysis or PCR.

51. The method of claim 48, wherein ABC1 expression is measured by protein analysis.

52. The method of claim 51, wherein said protein analysis is ELISA or Western blot.

53. The method of claim 48, wherein said cell comprises an exogenous marker cassette comprising a polynucleotide encoding a screenable marker operably linked to an ABC1 promoter region.

54. The method of claim 53, wherein the screenable marker is an esterase, phosphatase, protease, green flourescent protein, luciferase, chloramphenicol acetyl transferase, β-galactosidase, β-glucuronidase or a drug resistance marker.

55. The method of claim 48, wherein said cell expressing an RXR is an intestinal cell.

56. The method of claim 48, further comprising the step of determining the expression of ABC1 in a cell expressing RXR in the absence of said candidate substance.

57. The method of claim 48, wherein said screening for a modulator of ABC1 expression is performed *in vivo*.

58. A method of making a modulator of ABC1 expression comprising:

(a) providing a cell expressing an RXR;

(b) contacting said cell with a rexinoid and a candidate substance;

(c) determining the expression of ABC1 in said cell, wherein a change in expression of ABC1, as compared to a cell of step (b), indicates that said candidate substance is a modulator of ABC1 expression; and

(d) making said modulator.